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Clinically Silent Primary Adrenal Lymphoma: A Case Report and Review of the Literature

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Primary adrenal lymphoma (PAL) is extremely uncommon. We describe a case of clinically silent non-Hodgkin's B-cell lymphoma of diffuse large cell type with exclusive left adrenal localization. The tumor was discovered by computed tomography (CT) as a 2.5-cm dense mass and diagnosed at autopsy. Literature concerning this unusual neoplasm is reviewed. During the early stage, particularly when the lesion is small, PAL is likely to be missed. This unusual entity should be included in the differential diagnosis of adrenal masses so that early diagnosis may be made and intervention might dramatically affect the clinical outcome. Am. J. Hematol. 58:130–136, 1998. © 1998 Wiley-Liss, Inc.

Key words: adrenal gland; primary lymphoma; clinicopathologic features

INTRODUCTION

Secondary involvement of the adrenal glands by a malignant lymphoma has been reported to occur in as many as 25% of the patients with the disease [1,2]. In contrast, primary adrenal lymphoma is extremely uncommon and only 65 cases have been reported worldwide [3–45]. Because of its rarity, we report here an autopsied case of a patient with this unusual malignancy as well as review the literature regarding its clinical manifestations, histopathologic features, diagnosis, prognosis, management, and its possible pathogenesis.

CASE REPORT

A 46-year-old Hispanic male was admitted for a 3-day history of right foot swelling and right upper arm abscess with purulent drainage due to infection of the injection site. His past medical history is significant for diabetes mellitus, hypertension, liver cirrhosis due to hepatitis C infection and alcohol abuse (30 years), and intravenous drug abuse (IVDA) (20 years including left hand shooter's abscess with incision and drainage several years ago). He had not taken any medication for 6 months before admission. However, the patient had injected heroin the day prior to admission.

Physical examination revealed temperature of 98.5°F, pulse of 102/min, respiratory rate of 20/min, and blood pressure of 122/80 mmHg. Tachycardia with irregular rhythm and cardiac friction rub, 2^+ right foot edema and right deltoid abscess with spontaneous pustular drainage were also detected. There was no lymphadenopathy, organomegaly, or other significant findings.

Pertinent admitting laboratory results included leukocytosis (white blood cell 22.7×10^9/L) with neutrophilia and a left shift (segmented neutrophils 64%, bands 27%, metamyelocytes 2%, myelocyte 1%, lymphocytes 1%, and monocytes 5%). There was only mild derangement of blood chemistry and coagulation tests [sodium 130 meq/L (normal 135–145), potassium 5.5 meq/L (nl 3.5–5.0), chloride 95 meq/L (nl 98–107), bicarbonate 14 meq/L (nl 22–28), blood urea nitrogen 28 meq/dl (nl 8–20), creatinine 1.3 mg/dl (nl 0.6–1.2), prothrombin time 20 sec (nl 11–15), and activated partial thromboplastin time 38 sec (nl 25–35)] with the exception of a markedly elevated blood glucose of 505 mg/dl (nl 60–115) and lactic acid dehydrogenase of 836 U/L (nl 88–230). The serology test for human immunodeficiency virus-1 was negative. Electrocardiograph showed tachycar-

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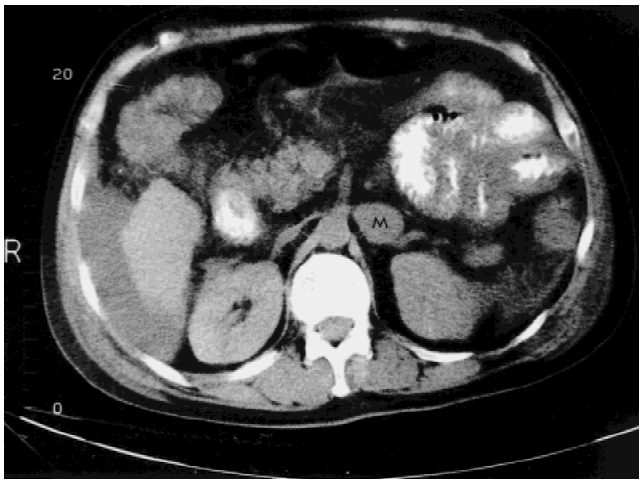

Fig. 1. Transverse upper abdominal CT scan image shows a round 2.5-cm. homogeneous mass (M) in the left adrenal gland.

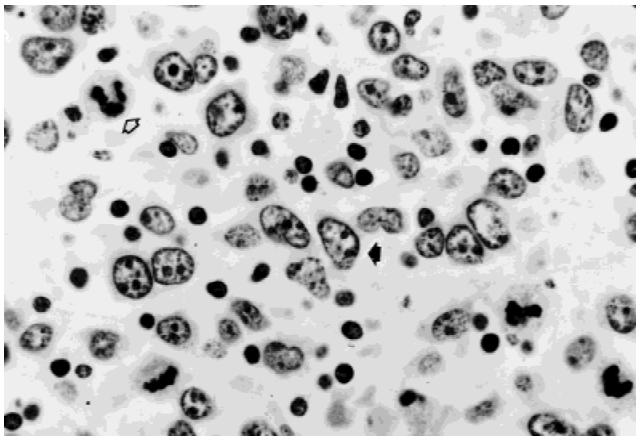

Fig. 2. Lymphoma cells have large, round and folded vesicular nuclei with one or multiple prominent nucleoli (arrow) and scant to moderate amount of cytoplasm. Mitotic figures (open arrow) are apparent (H & E, ×400).

dia with diffuse elevation of ST-segments and atrial fibrillation. Lower extremity X ray revealed right fibular periostitis and erosion of the second and third metatarsal heads. Ultrasound showed no evidence of lower extremity deep venous thrombosis. The clinical diagnoses of right foot osteomyelitis, right deltoid shooter's abscess and pericarditis were made. Both cultures of blood and abscess confirmed the presence of *Staphylococcus aureus* and *group B streptococci.*

The patient was treated with intravenous antibiotics (penicillin, oxacillin, cefotetan, and clindamycin) and other supportive managements. Echocardiography failed to reveal pericardial effusion or endocardial vegetation. In order to rule out other sources of infection, an abdominal/pelvic CT scan was performed that showed no evidence of intraabdominal/pelvic abscess or lymphadenopathy but a 2.5-cm rounded dense mass of the left adrenal gland was found (Fig. 1.). The right adrenal gland was normal. Cortisol stimulation test showed a baseline cortisol of 48 μg/dl, 30-min cortisol of 48.3 g/dl, and a 60-min cortisol of 54 μg/dl. The patient developed altered mental status, hypotension, and high fever (102°F) on hospital day 2, and was intubated and transferred to intensive care unit. Due to his deteriorative clinical situation, no further workup was done for the adrenal lesion. Right below knee amputation for the right dorsal foot abscess and incision and drainage of the right deltoid abscess were performed on hospital day 8. The patient had a complicated clinical course including sepsis, hypotension, acute renal failure, upper gastrointestinal bleeding and melena despite aggressive clinical treatment. The family desired not to resuscitate. The patient expired on hospital day 12. An autopsy was performed.

PATHOLOGIC FINDINGS

Major autopsy findings included diffuse fibrinous pericarditis, myocarditis with multiple intramyocardial abscesses, a large right deltoid abscess and left foot ulcer with gram positive cocci colonization, small focal pulmonary thromboemboli, bronchopneumonia, and macronodular cirrhosis of the liver.

An incidental finding was a primary lymphoma of the left adrenal gland, which weighed 14 g (nl 5–10). Cut sections revealed a poorly demarcated tan-gray lesion replacing the majority of the adrenal cortex and medulla. The lesion was grossly confined within the adrenal capsule. The right adrenal weighed 8 g and was normal in appearance.

Microscopically, a malignant lymphoma replaced most of the left adrenal gland. Neoplastic cells were large with pleomorphic vesicular nuclei and one to several large prominent nucleoli and a scant to moderate amount of basophilic cytoplasm (Fig. 2). Mitotic figures including abnormal ones were frequently seen. Scattered throughout the tumor were residual clusters and individual adrenal cortical and medullary cells, as well as scattered small ''mature'' lymphocytes. The tumor was microscopically confined within the adrenal gland. Upon immunohistochemical studies, these neoplastic cells expressed common leukocyte antigen CD45 and B-cell marker-CD20 but did not express T-cell markers CD43 and CD45RO. The tumor cells were negative for Epstein-Barr virus (EBV) latent membrane protein-1 (LMP-1) and EB encoded RNA (EBER). Examination of multiple lymph nodes from mediastinum, periaorta, and mesentery, as well as the spleen and bone marrow, failed to reveal any lymphomatous involvement.

DISCUSSION

Primary malignant lymphoma of the adrenal gland is extremely rare and so far only 30 cases [3–31] have been reported in the Western countries including France [3,26–29], Germany [30], Italy [24,31], The Netherlands [23], Spain [13,17,18,20,25], United Kingdom [10,11,19], and the United States [4–9,12,14–16,21,22]. There is one reported case from Taiwan [32], another from Hong Kong [33], and 33 other cases [34–45] from Japan over the past 49 years. The clinical and pathologic features of PAL have not been completely defined. We report here an additional case of clinically silent PAL in a Hispanic patient. We also review the literature of the reported cases.

The pertinent clinicopathologic features of the previously reported 55 cases, which include those in the English literature as well as those non-English reports containing enough information in the English abstracts, are summarized in Table I [3–16,18–23,28,30,32–35,37,39,41–45]. A more detailed account of each of the 30 isolated cases reported in the English literature is illustrated in Table II. PALs more commonly affect older men with a median age of 68 and a male to female ratio of 2.2 to 1. The majority of the reported cases (40/55) are bilateral, among which about 50% (20/40) manifest adrenocortical insufficiency even when the neoplasms are small (Table II case 8). Three of these cases presented as Addison's crisis [13,17,44]. It appears that there is no correlation between the size of tumor and adrenal dysfunction (Table II). Carey and associates [46] and Prayson and colleagues [47] reported one patient each who had angiotropic large cell lymphomas presented as adrenal insufficiency and Addison's crisis. We have not included these two cases in the series because of other organ involvement by lymphoma at the time of diagnosis. During the early stage of PAL, particularly when the lesion is unilateral and small, PAL is likely to be missed [44]. Autopsy may be necessary to differentiate primary PAL from those of systemic lymphomatous involvement. Our patient's clinical presentations and causes of death were unrelated to PAL. This was probably because the tumor was found in the early stage.

A provisional diagnosis of PAL could be made on the basis of the criteria described in the literature, which include the presence of adrenal mass, no evidence of superficial nodal involvement at first admission, and absence of a leukemic blood picture as well as other organ involvement [4,43]. The increasing usage and wide availability of ultrasonography (US), CT, and magnetic resonance imaging (MRI) have rendered a high number of incidentally discovered adrenal masses [13] and an increased awareness of PALs. Successful preoperative diagnoses of PALs with CT, MRI, and/or US-guided biopsies have been documented (Table II). Early diagnosis with percutaneous aspiration or core biopsy is necessary in order to begin therapy as soon as possible and avoid serious and potentially fatal complications [13,20].

TABLE I. Clinical Features in 55 Primary Adrenal Lymphoma Patients Reviewed in the Literature

General findings	
Male:female	38:17
Age (years)	
Median	68 (39–89)
Mean	65
Immune status	
Concurrent cancer[a] or past history of cancer[b] (4 each)	8 (15%)
HIV infection	2 (4%)
Autoimmune diseases[c]	7 (13%)
Symptoms, signs and other findings	
Fever	25 (46%)
Weight loss	13 (24%)
Pain [abdominal (9), other sites (5)]	14 (26%)
Skin pigmentation	8 (15%)
Adrenal insufficiency	20 (36%)
Definitive diagnostic procedure	
Needle aspiration or biopsy[d]	19 (35%)
Surgery (incisional biopsy or adrenalectomy)	16 (29%)
Autopsy	20 (36%)
Size and site of tumor	
Size (cm. in greatest diameter)	3–17
Site	
Bilateral	40 (73%)
Left	11 (20%)
Right	4 (7.3%)
Histopathology	
Large cell[e]	39 (71%)
Mixed large and small cell[f]	5 (9%)
Small cell[g]	6 (11%)
Lymphoma, NOS[h]	5 (9%)
Outcome	
Complete remission	7
Partial response	13
Death or unspecified	35
Duration of survival (diagnosis to death)	3 days to 26 months[i]

[a]Rectal adenocarcinoma (1), squamous cell carcinoma of scrotum (1), thyroid cancer (1), cholangiocarcinoma (1).
[b]Noninvasive duodenal carcinoma (1), prostatic carcinoma (1), uterine cancer (1), bladder carcinoma and laryngeal carcinoma (1).
[c]Systemic lupus erythematosus (1), hemolytic anemia (1), thrombocytopenia (1), diabetes mellitus (3), inappropriate ADH secretion (1).
[d]One patient had CT-guided Tru-cut biopsy; the other patient had histologic confirmation by bone marrow examination 3 months after initial diagnosis of bilateral PAL by CT and S/P chemotherapy.
[e]Including reticulum cell sarcoma, histiocytic lymphoma, pleomorphic large cell lymphoma, immunoblastic lymphoma, centroblastic cell lymphoma, and one CD30 positive anaplastic large cell lymphoma. All of the tumors that have been immunotyped expressed B-cell markers, except one case.
[f]One marked as T-cell.
[g]Including two small non-cleaved and one undifferentiated cell lymphoma.
[h]Including three cases classified as medium-sized B-cell.
[i]One patient survived 4 years but no further follow-up data are available.

TABLE II. Clinicopathologic Findings in 30 Patients With PAL Presented as Isolated Case Reports in the English Literature*

Reference, year	Case no.	Age/sex	Presenting symptoms	Immunostatus or concurrent disorders	Adrenal function	Diagnosis	Site	Size (cm in greatest diameter)	Pathology	Outcome
[8], 1961	1	56/F	Retrosternal pain	Diabetes	NM	IVP; autopsy	Bil.	L 15; R 12	Large and small cell lymphoma	No therapy; died 3 days later
[15], 1970	2	71/M	Anorexia, nausea, postural hypotension	Diabetes	Insufficiency	Autopsy	Bil.	Large but size and weight NS	Reticulum-cell sarcoma	Cortisone; died of pneumonia
[5], 1983	3	39/F	FUO	NM	Non-contributory	Sonography; angiogram, operation	L	Large but size and weight NS	Large cell NHL (histiocytic)	Surgery; NS
[6], 1983	4	45/M	Headache	Hypertension	NM	IVP; sonography; CT; operation	L	L 15	Immunoblastic B-cell sarcoma	Surgery; NS
[9], 1983	5	53/M	NM	NM	NM	CT; FNA	L	L 8	Undifferentiated lymphoma	NS
[14], 1985	6	81/M	Back pain	Glaucoma	Insufficiency	CT; sonography; FNA	Bil.	L 7.5; R 4.4	Large cell lymphoma	Cortisone; died 2 months later
[23], 1986	7	43/M	Abdominal pain, fever	Hypertension	NM	Sonography; CT; operation	Bil.	Large but size and weight NM	Diffuse histiocytic NHL	Surgery; chemotherapy; reduced tumor size on CT
[12], 1986	8	74/M	Fatigue, weight loss, fever	COPD, CAD	Insufficiency	CT, FNA, open biopsy	Bil.	L 3; R 4	Diffuse large cell (T-cell) lymphoma	Chemotherapy; died of sepsis and pneumonia
[16], 1987	9	68/M	Jaundice	NM	NM	Sonography; CT; operation	R	R 7	Immunoblastic B-cell NHL	Surgery; NS
[34], 1988	10	68/M	FUO	Interstitial pneumonitis, duodenal ulcer	NM	CT; autopsy	Bil.	L 7.4; R 8.1	Diffuse mixed cell (B-cell) lymphoma	Cortisone; pt became leukemic with dissemination; died 1 month later
[7], 1989	11	68/M	Jaundice	ASCVD; liver dysfunction; UTI	NM	Sonography; CT; operation	R	R 7	Large immunoblastic B cell	Surgery; chemotherapy; NS
[11], 1989	12	59/M	Weight loss, hyperpigmentation, nausea, vomiting	Anemia	Insufficiency	Sonography; CT-guided needle biopsy	Bil.	NM	Centroblastic cell NHL	Chemotherapy; died of aspergillosis
[32], 1989	13	68/F	Abdominal pain	NM	Normal	IVP; CT; sonography; operation	L	L 4	Lymphoma, NS	Surgery; chemotherapy; survived but duration NS
[33], 1990	14	52/M	Weight loss, fever	NM	NM	Sonography; CT; incisional biopsy	Bil.	L 7; R 8	CD30+ large cell lymphoma	Chemotherapy with a positive response
[10], 1990	15	59/M	Hyperpigmentation, weight loss, nausea, vomiting, nightsweats	NM	Insufficiency	Sonography; CT; Tru-cut biopsy	Bil.	L 15.8; R 10.2	Centroblastic cell NHL	Chemotherapy; died of aspergillosis
[4], 1990	16	64/M	Abdominal pain	Chronic bronchitis	Normal	Sonography; CT; operation	R	R 17	Large noncleaved B cell	Surgery; died at home

(*Continued*)

TABLE II. Clinicopathologic Findings in 30 Patients With PAL Presented as Isolated Case Reports in the English Literature (Continued)

[22], 1991	17	78/F	Abdominal pain	NM	Normal	Sonography; CT; incisional biopsy	L	L 7	Diffuse histiocytic NHL	Chemotherapy; good response
[19], 1992	18	68/M	Weight loss, fatigue, hyperpigmentation	NM	Insufficiency	CT; FNB	Bil.	L8; R 10	Centroblastic cell lymphoma	Cortisone; chemotherapy; radiation; in remission 9 months later
[39], 1992	19	72/F	Fatigue, fever and loss of consciousness	NM	Insufficiency	CT; angiogram; operation	Bil.	L 8.9; R 9.3 with liver, kidney and inferior vena cava invasion	Diffuse large B cell NHL	Cortisone; surgery; chemotherapy; died of multiliver recurrence 1 year later
[3], 1992	20	78/M	Abdominal pain, fever	HIV(+)	NM	Sonography; CT; operation	L	L 8	Centroblastic cell NHL	Chemotherapy; in remission 2 months later
[13], 1993	21	71/M	Nausea, weight loss, vomiting, weakness, fever	Hypotension	Insufficiency	Sonography; CT; FNB	Bil.	L 9; R 9.5	Small cleaved cell lymphoma	Chemotherapy; complete remission 4 months later
[18], 1993	22	59/F	Abdominal pain, weight loss, weakness	NM	NM	CT; FNB	Bil.	L 8.5; R 4	Pleomorphic large cell NHL	Chemotherapy; tumor regression on CT
	23	69/M	Same as above	NM	NM	CT; FNB	Bil.	L 5.1; R 5.3	Large cell NHL	Chemotherapy; tumor resolution on CT
[21], 1993	24	71/M	Weight loss, fatigue, diarrhea	NM	Insufficiency	CT; autopsy	Bil.	NM	Small noncleaved cell lymphoma	Early death from tumor
[20], 1995	25	68/F	Abdominal pain, lumbar pain, nausea	NM	Normal	CT; FNA	Bil.	L 10; R 7	Medium-sized B cell NHL	NS
[41], 1995	26	87/F	Lumbar pain, fever	NM	NM	CT: diagnosed on bone marrow examination	Bil.	Large but size and weight NM	Diffuse small non-cleaved cell NHL	Chemotherapy; reduced tumor size on CT; died of sepsis
	27	77/M	Malaise, anorexia	NM	NM	Sonography; CT; FNB	Bil.	Same as above	Diffuse medium-sized cleaved cell NHL	Chemotherapy; left testis met.; died of lung aspergillosis
	28	75/M	Anorexia	NM	NM	Sonography; CT; FNB	Bil.	Same as above	Same as case no. 25	Chemotherapy; in remission 4 years later
[44], 1996	29	60/M	Fatigue, hyperpigmentation, fever, shock	NM	Insufficiency	CT; autopsy	Bil.	L 7, R 7	Centroblastic cell NHL	Cortisone; died of lung infection 1 month later
[45], 1996	30	69/M	Abdominal pain, weight loss	NM	Normal	Sonography; CT; MRI; angiogram; FNB	Bil.	NM	Diffuse large B cell NHL	NS

*ASCVD, atherosclerotic cardiovascular disease; Bil, bilateral; L, left; R, right; CAD, coronary arterial disease; COPD, chronic obstructive pulmonary disease; CT, computed tomography; FNA, fine needle aspiration; FNB, fine needle biopsy; FUO, fever of unknown origin; HIV, human immunodeficiency virus-1; IVP, intravenous pyelogram; Met., metastasis; MRI, magnetic resonance imaging; NHL, non-Hodgkin's lymphoma; NM, not mentioned; NS, not specified; PAL, primary adrenal lymphoma; UTI, urinary tract infection.

On CT and MRI, PALs tend to appear as complex masses with variable desity; but a few cases also show a homogeneous density [18] and lesions with cystic appearance due to necrosis have been documented as well [5,6,10]. The masses are low-signal intensity on T1-weighted MR images and high-signal intensity on T2-weighted images [21,45], with occasional areas of mixed signal [21]. Angiographic appearance could be avascular [6] or hypovascular [23,45] but is not specific. Angiogram [5,6,7,23,39] and intravenous pyelogram [8,12,26] are useful to distinguish renal lesions from adrenal lesions. Nevertheless there is no pathognomonic appearance on CT, MRI, or US to indicate lymphomatous involvement of adrenal glands and one cannot distinguish primary from metastatic lesions [19]. The diagnosis of PALs in other cases is made by surgeries [3,5,6,7,12,16, 22,23,33,39] and postmortem examinations [8,15,34,41].

Morphologically, the most common type of lymphomas encountered in PALs is diffuse large cell, including centroblastic, immunoblastic, histiocytic, pleomorphic large cell, and one case of CD30 positive anaplastic large cell lymphoma (Table II). Other less common histologic types comprise mixed large and small cell, small noncleaved cell, undifferentiated, and others. It is interesting to note that the patient who had concurrent HIV infection had a centroblastic lymphoma [3], as did the AIDS patient who had a diffuse large cell lymphoma [43]. Based on the immunostaining, the majority of the reported cases are of B cell type with only two cases of T cell type documented so far [12,43]. PAL may be misdiagnosed as poorly differentiated carcinoma on aspiration [4,46]. However, a definitive diagnosis should not be difficult based on immunohistochemical and electron microscopic studies.

PAL is presumed to originate from hematopoietic tissue inherent to the adrenal gland [22,45]. So far all reported cases are of non-Hodgkin's lymphomas (NHL) [43]. Recently, EBV genome and LMP-1 were detected in lymphoma cells in 9/20 (45%) PAL cases by polymerase chain reaction, immunostaining, and/or in situ hybridization [43]. These findings implicate that EBV may be a possible causative agent in PAL similar to B-cell NHL in immunocompromised individuals. The prevalence of PAL in older individuals, especially those who had a history or concurrence of cancers, HIV infection, or autoimmune disorders, supports the notion that abnormal immune function or immunodeficiency may indeed play a role in the pathogenesis of PAL. In the present case, immunocompromise was possible due to drug abuse, infection, cirrhosis, and diabetes mellitus although his CD4 count was not done. Cytogenetic studies of two PAL patients also showed clonal abnormalities including 8q24 in one case and 14q32 in another, which were similar to those observed in nodal or extranodal B cell lymphoma [41,48,49].

The therapeutic modalities for PAL include surgery, combination chemotherapy, surgery followed by chemotherapy and/or radiation therapy, in addition to corticosteroid replacement [3,41,43]. Of those reports that provided follow-up, most patients died of tumor, intercurrent diseases, or infections within 1 year (Table II, [43]). However, 7 and 13 of these 55 patients achieved a complete [13,18,31,35,37,41,43] or partial [3,18,19,22,23,32, 33,39,41,42,43] remission, respectively (Table II). One of the patients was in remission 4 years after treatment (Table II, case 28). Radiation therapy appears to be ineffective [19].

The case reported here serves as a reminder that primary lymphoma of the adrenal gland may occur although the event is extremely rare. Primary adrenal lymphoma should be included in the differential diagnosis of a retroperitoneal or suprarenal mass, or in patients who present with adrenal insufficiency. Early diagnosis and intervention may dramatically affect the clinical outcome.

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